Marked Reversible ST-T Abnormalities Induced by Cardiac Compression From a Retrosternal Gastric Tube Used to Reconstruct the Esophagus After Tumor Resection

A Case of a Diabetic Patient and Mini-Review of 7 Reported Patients

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SUMMARY

A 69-year-old male patient with type 1 diabetes mellitus had been under treatment at our outpatient clinic since the age of 65. He had previously undergone surgery for esophageal cancer at the age of 55; the excised portion of the esophagus was replaced by a retrosternal gastric tube. He was admitted to our hospital for suspected pneumonia on April 8, 2004. An electrocardiogram (ECG) on admission showed marked ST depression in leads V1 and V2, and prominent negative T waves in leads I and aVL; however, the T waves unexpectedly flattened after 2 minutes and the ST depression resolved after about 4 hours. On January 7, 2005, we performed a deep breathing test to analyze the effects of movements of the thoracic wall and intrathoracic structures on the ECG. In this test, deep inspiration induced ST depression reaching 0.5 mV in leads V1 to V3; this resolved on switching to deep expiration. ECG changes together with chest computed tomography images supported the concept that the ST-T abnormalities were induced by cardiac compression caused by expansion of the gastric tube between the sternum and heart. We have reviewed 7 other similar reported cases. (Int Heart J 2006; 47: 475-482)

Key words: Electrocardiography, ST-T segment, Esophageal cancer, Deep breathing test, Chest computed tomography, Echocardiography, Diabetes mellitus, Retrosternal reconstruction, Gastric tube, Cardiac compression

ELECTROCARDIOGRAPHY (ECG) is a useful tool for detecting ischemic heart disease, particularly in diabetic patients, because they often lack typical symptoms suggesting myocardial ischemia. However, it has been pointed out that ECG abnormalities resembling myocardial ischemia may be induced by extracardiac factors.1,2) Recently, compression of the heart by a retrosternal gastric tube
used for esophageal reconstruction after resection of esophageal tumors has increasingly received attention as a new extracardiac factor that can induce prominent but reversible ECG abnormalities.\textsuperscript{3-8)} Resection of the esophagus followed by retrosternal reconstruction with a gastric tube is the most common surgical treatment for esophageal cancer in Japan. The diabetic patient presented in this report demonstrated marked reversible abnormalities in ECGs recorded 14 years after retrosternal gastric tube reconstruction was performed following resection of an esophageal tumor.

**CASE REPORT**

A 69-year-old male patient who had been under treatment for type 1 diabetes mellitus at our outpatient clinic since the age of 65 was admitted for suspected pneumonia on April 8, 2004. He had previously undergone retrosternal esophageal reconstruction using a gastric tube after excision of an esophageal tumor at the age of 55. He had never complained of any pain or discomfort in his chest that suggested ischemic heart disease. On admission, his chief complaints were cough and chills. His height was 166 cm and weight was 46.1 kg. He had a high fever of 39.9°C associated with a sinus tachycardia of 122/min. His blood pressure was 104/60 mmHg. Physical examination revealed nothing abnormal; respiratory and

![Figure 1. Electrocardiograms recorded on June 24, 2003 and April 8, 2004.](image-url)
heart sounds were normal. Laboratory examinations suggested acute inflammation; C-reactive protein was 7.75 mg/dL. Mild normocytic anemia and hyponatremia of 127 mEq/L were also evident; however, other laboratory findings were unremarkable except for hyperglycemia of 113 mg/dL and hemoglobin A1c of 9%. Antibiotics were administered intravenously, resulting in resolution of the fever after several days. The hyponatremia was normalized with intravenous saline solution.

Figure 1 shows an ECG recorded on June 24, 2003 and 3 ECGs recorded consecutively on the day of admission. The ST segments and T waves were unremarkable in the first ECG but appeared quite different on admission. Marked depression of ST segments in leads V1 and V2 and nearly symmetrical inversion of T waves in leads I and aVL were visible on the ECG recorded at 10:14 on the day of admission. Curiously, the T wave was clearly positive in lead V6, contrary to the generally accepted vector theory in which the T loop should be projected to almost the same direction in leads I and V6. Unexpectedly, the negative T waves in leads I and aVL appeared flattened when the ECG was repeated after 2 minutes. The depressed ST segments in leads V1 and V2 normalized after approximately 4 hours. Although the patient had not complained of chest pain, the series of ECG abnormalities could not exclude the possibility that the patient had ischemic heart disease. We therefore started isosorbide dinitrate administration.

In the follow-up period, ECGs were recorded on May 21, 2004, November 17, 2004, April 7, 2005, July 1, 2005, August 1, 2005, and November 24, 2005. Only 3 of these 6 demonstrated ST-T abnormalities. The patient complained of
chest pain on July 9, 2005, but an ECG recorded that same day showed no abnormalities. Neither beta-methyl-p-iodophenyl-pentadecanoic acid (BMIPP) nor thallium myocardial scintigraphy demonstrated any sign of myocardial ischemia. Fibergastroscopic examination revealed a gastric ulcer, which might have accounted for the chest pain. On January 7, 2005 we performed a deep breathing test to analyze the effects of movement of the thoracic wall and intrathoracic structures on the ECG. As shown in Figure 2, deep inspiration induced ST depression reaching 0.5 mV in leads V₁ and V₃; this normalized soon after switching to deep expiration. Similar ST changes during a deep breathing test were also recorded on September 8, 2005 and on December 6, 2005. ST-T abnormalities induced by postural changes were also reversible. For example, the ST-T abnormalities recorded in the right lateral position normalized immediately after switching to the left lateral position.

Figure 3 shows a computed tomography (CT) image obtained on April 9, 2004, one day after admission. The image, at the level of the fourth intercostal space, demonstrated food residue and air in the dilated gastric tube which was located between the sternum and heart. An ECG recorded immediately after the CT scan also showed ST depression in leads V₁ and V₂. A CT image and ECG that were similar were also recorded on April 7, 2005. A double Master's 2-step test on November 22, 2001 was negative. There were no findings that suggested

**Figure 3.** Chest CT image at the level of the fourth intercostal space on April 9, 2004. Arrows show large amount of food residue and air in the dilated gastric tube. Electrocardiogram recorded immediately after the CT scan shows ST depression in leads V₁ and V₂.
myocardial ischemia in the Holter electrocardiograms conducted on April 19, 2004 and on November 17, 2004. Other than a dilated gastric tube in front of the right ventricle, there were no abnormal findings in the echocardiograms performed on April 14, 2004 and on January 28, 2005. No findings suggested pulmonary thromboembolism. After following the present patient for approximately 19 months and reviewing case reports in which all ST-segment abnormalities except one were induced by cardiac compression from a reconstructed gastric tube, we concluded that the ST-T changes in this patient were induced by the same mechanism as that proposed in the case reports.

TITLE

Patients with diabetes mellitus have been known to develop silent myocardial ischemia in which ECG abnormalities suggesting myocardial ischemia are recorded without typical symptoms. ECG is therefore a very useful tool for early detection of silent myocardial ischemia. However, ECG changes resembling those of myocardial ischemia can also be induced by extracardiac factors. One of these factors is ketoacidosis, which is closely associated with diabetes mellitus. However, this was not observed in the present patient. In a retrospective analysis, the 3 consecutive ECGs recorded on admission showed the following unusual findings. Firstly, ST depression was observed only in leads V1 and V2. Secondly, nearly symmetric T-wave inversion was observed in leads I and aVL but not in V6. Thirdly, the negative T waves in I and aVL flattened in only 2 minutes. Considering these findings, we should have paid more attention to nonischemic causes of these ST changes. Although CT performed on the day after admission demonstrated dilation of the gastric tube between the sternum and heart, we could not exclude the possibility that the patient had ischemic heart disease.

In the 19-month follow-up period we noticed that the ST-T changes were not sustained in a series of ECGs and that they were reversibly induced by breathing or postural changes. These findings strongly suggest that the ST-T abnormalities were of nonischemic origin and were induced by movements of the thoracic wall and the intrathoracic structures. Both the ECG abnormalities and CT images are essentially compatible with those described in previous case reports in which ECG abnormalities were induced by a dilated gastric tube that had been sited for esophageal reconstruction.

In the first case report, Kamimura, et al described a 75-year-old male patient with marked ST-T changes after retrosternal reconstruction of the esophagus. An ECG recorded in the intensive care unit in the immediate postoperative period demonstrated significant elevation of ST segments in leads V1 to V4 and
slight depression of ST segments in leads II, III, and aVF. Despite continuous
administration of diltiazem and nitroglycerin these ST-T changes persisted, and
tended to become more pronounced during expiration and less obvious during
inspiration. Coughing 7.5 hours later expelled a large amount of air from the drain
inserted into the gastric tube. This was followed by complete resolution of the ST-
T changes, which were not observed again despite frequent follow-up ECGs dur-
ing a 5-day stay in the intensive care unit. However, that case differed from our
own in several ways. Firstly, the ECG abnormalities appeared immediately after
esophageal reconstruction in the patient of Kamimura and were first recorded 14
years after a similar operation in the present patient. Moreover, an appropriate
comparison is difficult, because the observation periods differed considerably
between the 2 patients. The patient described by Kamimura, et al was observed
for only 5 days in the immediate postoperative period, while our patient was
observed for a much longer period of 19 months, from 14 years after surgery. Sec-
ondly, the ECG changes also differed between the patients. Marked elevation of
ST segments reaching over 2 mV in lead V2 without visible changes in T waves
was demonstrated in their patient, in contrast to clear depression of ST segments
in leads V1 and V2 with inversion of T waves in leads I and aVL in the present
patient. Differences in the spatial relationship between the dilated gastric tube
and heart might be responsible for these differences in ECG abnormalities.
Thirdly, the ST segment elevation increased during expiration in their patient, but
ST segment depression was induced during inspiration in the present patient. This
difference was also attributed to individual complex movements of the heart and
other thoracic structures induced by breathing in each patient. The ECG changes
disappeared after air was expelled from the gastric tube in their patient, and
although the ECG normalized within 2 minutes in the present patient, we did not
confirm expulsion of air from the dilated gastric tube.

The Table presents a list of 7 reported patients in whom ECG changes
appeared after esophageal reconstruction for the treatment of cancer.3-8) Retrosternal
gastric tubes were used for esophageal reconstruction in all patients except
one.5) Three of the 7 patients complained of chest pain or oppression. In all
patients, the main ECG changes were elevation or depression of the ST segment.
T wave changes were not described in any of these 7 patients, although such
changes were recorded in the present patient. The ECG abnormalities were attrib-
uted to cardiac compression by the dilated gastric tube in 6 patients and to coro-
nary vasospasm in one patient.4)

In conclusion, we have described a diabetic patient who showed marked
reversible ST-T abnormalities 14 years after retrosternal gastric tube reconstruc-
tion for the treatment of esophageal cancer. ECG changes together with chest
computed tomography images supported the concept that the ST-T abnormalities
were induced by cardiac compression caused by expansion of the gastric tube between the sternum and heart.

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REFERENCES